

HHS Public Access

Author manuscript

Pediatrics. Author manuscript; available in PMC 2018 April 09.

Published in final edited form as:

Pediatrics. 2016 November; 138(Suppl 1): S3-S14. doi:10.1542/peds.2015-4268C.

Opportunities During Early Life for Cancer Prevention: Highlights From a Series of Virtual Meetings With Experts

Dawn M. Holman, MPH and Natasha D. Buchanan, PhD on behalf of the Cancer Prevention During Early Life Expert Group

Division of Cancer Prevention and Control, Centers for Disease Control and Prevention, Atlanta, Georgia

Abstract

Compelling evidence suggests that early life exposures can affect lifetime cancer risk. In 2014, the Centers for Disease Control and Prevention's (CDC's) Cancer Prevention Across the Lifespan Workgroup hosted a series of virtual meetings with select experts to discuss the state of the evidence linking factors during the prenatal period and early childhood to subsequent risk of both pediatric and adult cancers. In this article, we present the results from a qualitative analysis of the meeting transcripts and summarize themes that emerged from our discussions with meeting participants. Themes included the state of the evidence linking early life factors to cancer risk, research gaps and challenges, the level of evidence needed to support taking public health action, and the challenges of communicating complex, and sometimes conflicting, scientific findings to the public. Opportunities for collaboration among public health agencies and other stakeholders were identified during these discussions. Potential next steps for the CDC and its partners included advancing and building upon epidemiology and surveillance work, developing and using evidence from multiple sources to inform decision-making, disseminating and communicating research findings in a clear and effective way, and expanding collaborations with grantees and other partners. As the science on early life factors and cancer risk continues to evolve, there are opportunities for collaboration to translate science into actionable public health practice.

The Healthy People 2020 goal for cancer is to reduce the number of new cancer cases, as well as the illness, disability, and death caused by cancer. ¹ Incidence rates have increased over the past decade for certain types of childhood cancers (eg, leukemia and renal carcinomas) ² and adult cancers (eg, melanoma, myeloma, leukemia, and cancers of the

Address correspondence to Dawn M. Holman, MPH, Division of Cancer Prevention and Control, Centers for Disease Control and Prevention, 4770 Buford Hwy NE, Mailstop F76, Atlanta, GA 30327. dholman@cdc.gov.

The findings and conclusions in this report are those of the authors and do not necessarily represent the official position of the Centers for Disease Control and Prevention.

FINANCIAL DISCLOSURE: The authors have indicated they have no financial relationships relevant to this article to disclose. **POTENTIAL CONFLICT OF INTEREST**: The authors have indicated they have no potential conflicts of interest to disclose.

Ms Holman conceptualized and designed the study, conducted qualitative analysis of the meeting transcripts, contributed to the interpretation of the qualitative data, drafted sections of the initial manuscript, and critically reviewed and revised the manuscript; Dr Buchanan developed the initial strategy qualitative data analysis, conducted qualitative analysis of the meeting transcripts, contributed to the interpretation of the qualitative data, drafted sections of the initial manuscript, and critically reviewed and revised the manuscript; each member of the Cancer Prevention During Early Life Expert Workgroup provided substantial input on the content and agendas for the virtual meeting series and critically reviewed and revised the manuscript; and all authors approved the final manuscript as submitted and agree to be accountable for all aspects of the work.

pancreas, liver, thyroid, and kidney). ³ Although cancer incidence rates are expected to stabilize in the coming years for most groups within the US population, the overall number of prevalent cancer cases and deaths will likely increase as a result of demographic changes (eg, a growing and aging population). ^{4,5} Public health approaches to addressing "upstream" causes of cancer continue to be a critical component of national efforts to prevent the onset of cancer at all ages.

The Cancer Prevention Across the Lifespan (CPAL) workgroup was organized within the Centers for Disease Control and Prevention's (CDC's) Division of Cancer Prevention and Control in an effort to foster innovative public health approaches to primary cancer prevention. ⁶ Grounded in a socioecological framework, ⁷ workgroup activities include reviewing the scientific literature and engaging with experts both within and outside of federal agencies to identify opportunities to reduce cancer risk and promote protective factors at a population level. The workgroup has taken a life-span approach, with the idea that both the exposures of interest and the strategies for intervention vary depending on the phase of life being addressed. In addition, this approach recognizes the complexities of translating the science of cancer causation into public health action and the value of transdisciplinary perspectives. ⁸ To date, the workgroup has published information from efforts to identify prevention opportunities during adolescence ⁹ and midlife, ¹⁰ with an ultimate goal of addressing cancer prevention opportunities during every phase of life.

Compelling evidence suggests that exposures during early life (the prenatal period through early childhood) can affect lifetime cancer risk. ^{11–16} In 2013, the workgroup began examining the influence that early life factors may have on subsequent risk of both childhood and adult cancers. As part of this effort, the CPAL workgroup convened a series of virtual meetings with a group of nationally recognized experts to explore the state of the evidence linking early life factors to subsequent cancer risk and to identify promising strategies for prevention with a focus on environmental and systems approaches. In this article, the results from a qualitative analysis of the meeting transcripts are presented, summarizing themes that emerged from these discussions.

METHODS

The CPAL workgroup hosted a series of five 2-hour online meetings to examine opportunities during early life to reduce the subsequent risk of cancer in childhood and adulthood. Five subject matter experts external to the CDC were invited to participate in each of the first 4 meetings, resulting in a multidisciplinary group of 20 experts participating in the meeting series. The participants were nationally recognized authorities on a diverse range of relevant topics, reflecting many areas of expertise and scientific discipline and with unique knowledge about early life. We intentionally reached out to experts both within and outside of traditional areas of cancer prevention and control in an effort to gain new perspectives on the potential opportunities for primary cancer prevention and risk reduction.

Before each meeting, the invited experts participated in developing the meeting agenda and identifying key background materials, all of which were shared with the group in advance of each meeting. During the meetings, each invited expert led the group in a discussion about a

specific topic relevant to the scope of the meeting as a whole, with a professional meeting facilitator guiding each discussion. The first meeting, held on May 23, 2014, focused on opportunities during the prenatal period to reduce the risk of childhood cancer. The second meeting (July 29, 2014) explored opportunities during the prenatal period to reduce the risk of cancer in adulthood. The third meeting (October 3, 2014) examined opportunities during infancy and early childhood to reduce the risk of childhood cancer. The fourth meeting (December 17, 2014) addressed opportunities during infancy and early childhood to reduce the risk of cancer in adulthood. After the completion of the first 4 meetings, 11 of the 20 experts accepted an invitation to participate in a follow-up meeting (March 13, 2015) to review and provide input on the preliminary results of a qualitative analysis of the transcripts from the previous 4 meetings and to discuss potential next steps and action items. The 5 meetings were recorded and transcribed with the permission of all participants.

ANALYSIS AND SYNTHESIS OF MEETING THEMES

A thematic content analysis of the meeting transcripts was conducted. A priori coding categories were developed to address 3 main questions (Table 1):

- **1.** What is important?
- **2.** What is missing?
- **3.** What can be done now?

These overarching questions and questions related to each subcategory were provided to participants in advance of and during each of the 5 meetings, making it likely that much of the content would fit into these predetermined categories. Content that did not clearly fit one of the categories was coded as "other." Two reviewers (DMH and NB) who were trained in qualitative thematic analysis independently coded the meeting transcripts using QSR International's NVivo 10 software (QSR International, Doncaster, Australia). Intercoder reliability was high, with agreement of >80% for each category. An emergent coding strategy was then used to identify and summarize themes within each category. Any discrepancies in coding were discussed and resolved.

THEMES AND HIGHLIGHTS FROM THE MEETINGS

Early Life Factors Associated With Cancer Risk

Experts described the causes of both childhood and adult cancers as being multivariate, multifactorial, and multigenerational, involving genetic and environmental interactions. Many specific examples of factors during early life that may influence subsequent cancer risk were discussed during the meetings. Potential risk factors discussed included adverse childhood experiences, certain parental behaviors (eg, alcohol consumption, tobacco use), chemical exposures (eg, occupational exposures, residential pesticide exposure), medication or drug exposure during pregnancy (eg, diethylstilbestrol), chromosomal abnormalities (eg, Down syndrome), genetic syndromes (eg, multiple endocrine neoplasia syndrome), older parental age, placental phenotype, characteristics of pubertal events (eg, early menarche), radiation exposure (eg, diagnostic radiographs in utero, nuclear fallout, radiation therapy), viral infections (eg, human papillomavirus), bacterial infections (eg, *Helicobacter pylori*),

and weight-related factors (eg, maternal prepregnancy weight, high birth weight, childhood obesity). Experts also identified some potentially protective factors, including allergies, asthma, atopy, dietary factors (eg, breastfeeding, prenatal folic acid consumption), early stimulation of the immune system (eg, day care attendance at an earlier age), and childhood physical activity. A more extensive list of examples discussed during the meeting is provided in Table 2, along with relevant supporting citations. At each meeting, the experts emphasized that the factors discussed were not exhaustive and that the strength and consistency of the evidence for each factor vary.

Current Challenges

Research Gaps—Experts agreed that the evidence linking some early life factors to subsequent cancer risk is strong and consistent across studies (eg, radiation exposure). For the majority of factors described above and in Table 2, however, the link to cancer risk is not as clear and the strength of the evidence varies, depending on the factor and the type of cancer being considered. Research findings may be mixed across studies, or the measures of relative risk may be small or not statistically significant. Experts also noted that there is often a lack of information or understanding about etiologic mechanisms that could explain research observations. A life-course approach acknowledges that countless factors throughout the life span likely influence cancer risk later in life and that certain factors may interact to influence cancer risk. The complex nature of these relationships inherently makes it difficult to accurately determine the influence of a single factor during 1 phase of life. Many cancer types, especially childhood cancers (in comparison with cancers diagnosed in adulthood), tend to be rare. Experts discussed how this rarity makes research more challenging and often limits human data to observational studies and retrospective studies, rather than prospective cohort studies which can sometimes yield stronger evidence on exposures. The retrospective nature of the available evidence poses the risk for a number of biases, especially recall and selection bias, although prospective studies can also be subject to limitations (eg., small sample sizes and selection bias). The interpretation of study findings needs to take into account the potential for these errors. As the research community gains new opportunities to collect more data, new methods are needed to analyze large data sets and to assess nonlinear, nonmonotonic relationships between exposure and disease. In addition, for most exposures of interest, there is a lack of viable biomarkers to measure dose or intermediate outcomes of interest.

Evidence Needed to Support Taking Action—Given the research gaps and existing challenges, many meeting discussions focused on how much evidence is needed to warrant taking public health action. This question is an ongoing challenge that scientists, public health professionals, and decision-makers face. Although there would be benefit to having more prospective and large human studies, some of the experts argued for a precautionary approach, ^{86, 87} explaining that animal studies and toxicologic profiles ⁸⁸ that suggest carcinogenicity would be "enough" to support intervention efforts. Others commented that human data are needed to warrant the investment of public health resources into prevention efforts. The experts pointed to systematic, structured reviews as a way to integrate the evidence from multiple sources and better synthesize the existing evidence. ^{89–91} Such

reviews need to be well designed to take into account any methodologic concerns surrounding the topic and the heterogeneity across studies. 92

Beyond the strength of the scientific evidence linking a factor to subsequent cancer risk, participants discussed many other aspects that may influence decisions to take (or not take) public health action. For example, experts noted that the prevalence of the given risk factor and the association (positive or negative) with other health outcomes ¹⁵ are important to consider (eg, the inverse association between birth weight and risk of cardiovascular disease ⁹³). When deciding whether to implement specific intervention or prevention strategies, both the short- and long-term effects, including potential unintended consequences or drawbacks to taking action (eg, harmful effects on other health outcomes), need to be considered. Experts also noted the importance of considering economic factors, including the cost of intervention, potential impact on industry, and the future costs of failing to intervene. ⁹⁴ The ease or difficulty with which an intervention strategy could be implemented is often very influential in decision-making. For example, removing potentially harmful chemicals from consumer products necessitates that safer alternatives are available. In addition, experts offered that the larger social context needs to be considered to determine the feasibility and sustainability of a given intervention.

Intervening on Harmful Chemicals—The experts also discussed the challenges communities may face in trying to reduce or remove chemical exposures when research findings suggest that they may be harmful to human health. Experts identified the lack of requirements for premarket safety testing of chemicals, frequent reliance on toxicologic studies with sparse human data, lack of safer alternatives, and low public awareness of exposures to industrial chemicals as potential barriers to intervening. Although the experts acknowledged the potential for industry resistance to removing chemicals currently in use, they also noted that there can be ways to incentivize and facilitate industry changes. For example, in Massachusetts, industry use of certain carcinogens declined after initiation of requirements that companies develop plans to reduce the use of toxic chemicals. 95, 96 Of note, the implementation of the plan was not required. This approach may be worth considering in other communities. Another challenge that experts pointed out is that regulatory interventions sometimes fail to address key windows of vulnerability. One example mentioned was the removal of bisphenol A (BPA) from infant bottles. Although likely reducing infants' BPA exposure, such action does not prevent prenatal BPA exposure. ⁹⁷ Of additional concern, replacements for chemicals of concern are not always safer. For example, many BPA-free replacement products still leach chemicals that have high levels of estrogenic activity. 98 The rapid industry changes that come with technological improvements also create challenges. For example, in California, researchers rely on existing data collection systems (eg, community-based air-monitoring stations), ⁹⁹ but as pollution emissions change over time these systems do not necessarily collect data on all of the relevant air pollutants.

Communicating Scientific Findings to the Public—Many of the challenges the research community faces create subsequent difficulties in sharing information with the public. Experts acknowledged that research findings are often complex or even conflicting,

making it difficult to create simple, compelling, and realistic recommendations for risk reduction. Some cancer risk factors may have a different relationship with other noncancer health outcomes. For example, high birth weight appears to be associated with an increased risk of certain pediatric and adult cancers, ^{22,26} but evidence also suggests that high birth weight may also be associated with a reduced risk of heart disease. ⁹³

Scientific understanding of cancer risk factors has evolved over time. Experts shared that when contradictory study findings are published or when health recommendations change, credibility with the public may be weakened. To communicate which risk factors matter most, measures of attributable risk are often presented. However, some noted that such measures have limited real-world utility and do not capture information such as windows of susceptibility (eg, the prenatal period), interactive effects of multiple factors, and the influence of protective factors (eg, physical activity). Competing demands, other more immediate health threats that children often face (eg, injuries, acute infections), and information overload can create barriers to effective messaging, making attention to the many potential cancer risk factors seem impractical, if not impossible, for the public to maintain. Experts also offered that some messages may unintentionally elicit public perceptions of "blaming the victim," increase cancer-related distress, or contribute to risky behaviors.

Suggested Next Steps for Interdisciplinary Stakeholders

Despite the challenges of understanding the influence that early life factors may have on subsequent cancer risk and communicating that information to the public, experts identified public health actions to further build the evidence base and put current information into public health practice. These actions include strengthening cancer epidemiology and surveillance, building upon existing information, disseminating and communicating research findings, and applying our current knowledge to public health practice through multidisciplinary partnerships.

Epidemiology and Surveillance—Together, CDC's National Program of Cancer Registries and the National Cancer Institute's Surveillance, Epidemiology, and End Results Program collect data on new cancer cases for the entire US population. ¹⁰⁰ This national coverage enables researchers, clinicians, policy makers, public health professionals, and members of the public to monitor the incidence of cancer and subsequently evaluate the success of programs and identify additional needs for cancer prevention and control efforts at national, state, and local levels. Meeting participants suggested that studies could integrate cancer registry data with other data sources, such as the NHANES, which collects biomarkers of exposure to carcinogens (eg, serum 1-hydroxypyrene, a biomarker for exposure to polycyclic aromatic hydrocarbons). ¹⁰¹ Such efforts could leverage the research methods used by others, such as efforts in California to examine the relationship between prenatal exposure to traffic-related air pollution and risk of childhood cancer. ⁴⁹ Experts expressed a need to study more finely detailed data such as cancer subtypes and cancer type-specific staging in cancer registries to better define cancer cases, which might provide additional clues about etiology. Experts also suggested looking to disease patterns outside of the United States for clues and considering both birth cohort effects and time period effects.

¹⁰² Adding to the list of biomarkers of exposure to carcinogens collected through NHANES to assess exposure in the general population, ensuring optimal biomarkers are used, ¹⁰³ and developing new biomarker tools (eg, epigenetic markers of in utero exposures) would facilitate further study of exposures of potential concern. ¹⁰⁴

Developing and Using Evidence From Multiple Sources—Additional steps are needed to take advantage of existing data and to continue building on the evidence. One suggestion is to continue conducting structured, systematic reviews of the published literature on both animal and human studies that, in addition to informing the current state of the evidence, will also help identify key research gaps. Convening multidisciplinary expert groups can help to frame which risk factors should be given priority and identify ways to address research gaps. Some health groups, such as the Intergovernmental Panel on Climate Change, ¹⁰⁵ have used a Bayesian approach to translate uncertain evidence into probabilistic statements. Such an approach could be used in assessing the cancer literature to create statements about the likelihood of an exposure causing cancer. Another suggestion was to create a Web site in which investigators could share study results, creating a cumulative summary of the evidence over time.

Disseminating and Communicating Research Findings—Despite varying views on the amount of evidence needed to take action and acknowledgment of the complexity of research findings, communication of scientific findings needs to be clear, accurate, easy to understand, and actionable. ¹⁰⁶ This method of communication equips community leaders, decision-makers, and the public with the information needed to make decisions about the appropriate threshold for action. Experts mentioned that communication and dissemination of research findings to the public could be improved by considering strategies for translation at the onset of developing research plans. For example, studies can be designed to examine the association between a given factor and cancer risk in a way that facilitates communicating the findings to the public and informs potential intervention strategies. Packaging cancer prevention messages with messages related to other health topics may provide new opportunities for collaboration and synergy. Communication about cancer prevention could be enhanced by including information about other health benefits (eg, reducing risk of other chronic diseases), the economic benefits, and the potential costs of not taking action. With regard to industrial chemicals, participants noted that there can be benefits to framing efforts to remove harmful chemicals from consumer products as cancer prevention, acknowledging that companies may prefer to remove specific chemicals without public attention or acknowledging the potential for past harms.

Working With Grantees and Other Partners—Creating partnerships, engaging in transdisciplinary collaboration, and learning from others are critical to the success of efforts to reduce cancer risk at a population level. The experts noted that many relevant public health organizations and agencies have overlapping interests and would benefit from collaboration. Examples of suggestions included continuing to foster partnerships between the CDC and the Institute of Medicine, the World Health Organization, the National Institutes of Health, and the International Society for Developmental Origins of Health and Disease. Exploring opportunities for new partnerships with various community sectors can

also be of value. For example, recent efforts in Massachusetts to incorporate asthma prevention into the state's Asthma Control Plan brought together a wide range of partners, including traffic and transportation planners. ¹⁰⁷ The CDC funds and provides technical support to Comprehensive Cancer Control programs in all 50 states, the District of Columbia, 7 tribes and tribal organizations, and 7 US territories. ¹⁰⁸ Many of these programs already address cancer risk factors in their communities and would likely benefit from a similar approach to developing partnerships with various community sectors.

Other specific areas that would benefit from continued public health efforts and partnerships to support change at the local, state, or national levels were mentioned by the experts during the meetings. For example, ongoing public health approaches are needed to increase human papillomavirus vaccination rates. ¹⁰⁹ Occupational benzene standards and the levels of enforcement need to be examined. 110,111 With increases in exposure to radiation from medical procedures in the United States, additional efforts are needed to identify strategies and develop partnerships for reducing exposure to unnecessary levels of radiation from medical imaging procedures. 112 There may also be opportunities to identify and develop interventions specifically for those at high risk of certain cancers in an effort to change their risk trajectory over time. For example, risk reduction and health promotion strategies may be particularly beneficial for those who experienced prolonged adversity or harmful exposures during early life or who have a family history of cancer. 113 Genetic testing policies were mentioned as another example and were described as an emerging area in which the CDC may have an opportunity to contribute. When considering such policies, it will be important to address how and when to communicate genetic risk in a way that minimizes potential harms and unintended consequences (eg, psychological distress) and to promote appropriate and effective interventions to support risk reduction. ^{114,115}

Continuing to Research, Learn, and Find Ways to Intervene Early—There are many emerging areas of research that may give additional clues for prevention. Some areas of interest mentioned by experts included the following: the role of the placental microbiome, associations of earlier pubertal measures with cancer risk, interactions between genetic risk factors and exposures to endocrine active substances, and the influence of contextual factors (eg, poverty, the family environment, neighborhood crime) and the biopsychosocial pathways ¹¹⁶ in which these factors influence disease. As the research community continues to strive to understand the complex causes of cancer and the influence of early life, compelling evidence of the benefit of intervening early already exists and needs to be translated into actions. This evidence may be particularly relevant in the context of children of cancer survivors who may be at an increased risk of a second cancer in adulthood because of genetic factors. By identifying children at an increased risk and providing both social support and support for healthy behaviors, we may be able to modify and reduce their cancer risk. Similarly, earlier exposure to protective factors (eg, physical activity) may have a greater protective effect, ¹¹⁷ suggesting that interventions in early life can change an individual's lifetime cancer risk trajectory.

CONCLUDING REMARKS

The scientific evidence about the influence of early life factors on cancer risk continues to evolve. Discussions during the virtual meeting series highlighted opportunities to leverage existing public health partnerships and approaches to optimize early life health and reduce lifetime cancer risk. Clear and direct messages that use current evidence-based knowledge could be more routinely shared with the public, taking careful consideration not to cause unintentional harms. Health messages related to cancer prevention could be combined with other health and economic information to assist community leaders in decisions about whether to take action (ie, implementing policy, system, or environmental changes). Developing new partnerships and building on existing relationships can help maximize synergy and success.

The CDC is already engaged in activities that could be used to advance cancer prevention efforts targeting early life, from research to implementation of prevention strategies. For example, the CDC's National Center for Health Statistics developed the Research Data Center (RDC) to allow researchers access to restricted data. ¹¹⁸ The RDC hosts restricted data from a variety of government sources, including NHANES, the National Health Care Surveys, the National Health Interview Survey, the National Vital Statistics System, the National Survey of Family Growth, National Program of Cancer Registries, and the State and Local Area Integrated Telephone Survey. The CDC has recently made restricted cancer registry data available through the RDC ¹¹⁹ and is evaluating federal, state, and local government policies regarding cancer research to promote the use of cancer registry data for research. These activities and data resources can be used in future research efforts related to early life exposures and cancer risk. In addition, the CDC has funded state cancer registries to enhance the tracking of pediatric and young adult cancer cases, support more timely reporting of these cases, and increase the availability of the corresponding surveillance data at the national, state, and local levels. 120 In an effort to communicate information about opportunities for cancer prevention at the community level, the CPAL workgroup has started a suite of materials on promising policies and practices, with an emphasis on ways in which CDC-funded Comprehensive Cancer Control programs can take actions in their communities. 121

Each phase of life has unique characteristics that warrant different approaches and strategies for reducing cancer risk. Factors during the prenatal period, infancy, early childhood, and even before conception appear to influence cancer risk, and intervening on risk factors early in life while promoting protective factors may be particularly beneficial. Although the science on this issue continues to evolve, there are opportunities now to work together to act on what is already known.

Acknowledgments

The Cancer Prevention During Early Life Expert Group includes the following members: Frank Biro, MD (Department of Pediatrics, University of Cincinnati College of Medicine; Division of Adolescent Medicine, Cincinnati Children's Hospital Medical Center); Angela Bradbury, MD (University of Pennsylvania Perelman School of Medicine; Department of Medical Ethics and Health Policy, University of Pennsylvania; Department of Medicine, Division of Hematology-Oncology, University of Pennsylvania); Dana C. Dolinoy, PhD (Department of Environmental Health Sciences, School of Public Health, University of Michigan); Terence Dwyer, MD, MBBS,

MPH (The George Institute for Global Health, University of Oxford; James Martin Fellow, Oxford Martin School, University of Oxford; Professor of Epidemiology, University of Oxford); Michele R. Forman, PhD (Department of Nutritional Sciences, College of Natural Sciences, University of Texas at Austin); Lynn R. Goldman, MD, MS, MPH (Milken Institute School of Public Health, George Washington University); Julia E. Heck, PhD (Department of Epidemiology, Fielding School of Public Health, University of California, Los Angeles); David Kriebel, ScD (Department of Work Environment, College of Health Sciences, University of Massachusetts Lowell); Catherine Metayer, MD, PhD (School of Public Health, University of California, Berkeley); Karin B. Michels, ScD, PhD (Obstetrics and Gynecology Epidemiology Center, Department of Obstetrics, Gynecology, and Reproductive Biology, Brigham and Women's Hospital, Harvard Medical School; Department of Epidemiology, Harvard School of Public Health); Andrew Olshan, PhD (Department of Epidemiology, University of North Carolina, Chapel Hill); C. Mary Schooling, PhD (School of Urban Public Health at Hunter College and City University of New York School of Public Health); Martyn Smith, PhD (School of Public Health, Division of Environmental Health Sciences, University of California, Berkeley); Logan G. Spector, PhD (Division of Epidemiology/Clinical Research, Department of Pediatrics, University of Minnesota); Mary Beth Terry, PhD (Department of Epidemiology, Columbia University Mailman School of Public Health; Herbert Irving Comprehensive Cancer Center, Columbia University Medical Center); Kent Thornburg, PhD (Oregon Health and Science University, Heart Research Center); Leonardo Trasande, MD, MPP (Departments of Pediatrics, Environmental Medicine and Population Health, New York University School of Medicine); Rebecca Troisi, MA, ScD (Division of Cancer Epidemiology and Genetics, National Cancer Institute, National Institutes of Health, Department of Health and Human Services); Tracey Woodruff, PhD, MPH (Program on Reproductive Health and the Environment, Department of Obstetrics, Gynecology and Reproductive Sciences, University of California, San Francisco); and Natasha D. Buchanan, PhD, Donatus Ekwueme, PhD, Violanda Grigorescu, MD, MSPH, S. Jane Henley, MSPH, Dawn M. Holman, MPH, Jun Li, MD, PhD, MPH, Greta M. Massetti, PhD, Mary Elizabeth O'Neil, MPH, Lucy A. Peipins, PhD, Mary Puckett, PhD, Meredith L. Shoemaker, MPH, Katrina F. Trivers, PhD, MSPH, and Mary C. White, ScD (The Cancer Prevention Across the Lifespan Workgroup, Division of Cancer Prevention and Control, Centers for Disease Control and Prevention, Department of Health and Human Services).

FUNDING: Ms Holman and Dr Buchanan are federal government employees and preparation of this manuscript was entirely funded by the US government.

ABBREVIATIONS

BPA bisphenol A

CDC Centers for Disease Control and Prevention

CPAL Cancer Prevention Across the Lifespan

RDC Research Data Center

References

- US Department of Health and Human Services. [Accessed August 11, 2015] Healthy People 2020 topics & objectives: cancer. Published 2015. Available at: www.healthypeople.gov/2020/topics-objectives/topic/cancer
- 2. Siegel DA, King J, Tai E, Buchanan N, Ajani UA, Li J. Cancer incidence rates and trends among children and adolescents in the United States, 2001–2009. Pediatrics. 2014; 134(4) Available at: www.pediatrics.org/cgi/content/full/134/4/e945.
- 3. Kohler BA, Sherman RL, Howlader N, et al. Annual report to the nation on the status of cancer, 1975–2011, featuring incidence of breast cancer subtypes by race/ethnicity, poverty, and state. J Natl Cancer Inst. 2015; 107(6):djv048. [PubMed: 25825511]
- Weir HK, Thompson TD, Soman A, M
 øller B, Leadbetter S. The past, present, and future of cancer incidence in the United States: 1975 through 2020. Cancer. 2015; 121(11):1827–1837. [PubMed: 25649671]
- 5. Weir HK, Thompson TD, Soman A, Møller B, Leadbetter S, White MC. Meeting the Healthy People 2020 objectives to reduce cancer mortality. Prev Chronic Dis. 2015; 12:E104. [PubMed: 26133647]

 Shoemaker ML, Holman DM, Henley SJ, White MC. News from CDC: applying a life course approach to primary cancer prevention. Transl Behav Med. 2015; 5(2):131–133. [PubMed: 26029275]

- Institute of Medicine (US) Committee on Assuring the Health of the Public in the 21st Century. The
 Future of the Public's Health in the 21st Century. Washington, DC: National Academies Press;
 2002.
- 8. White MC, Peipins LA, Watson M, Trivers KF, Holman DM, Rodriguez JL. Cancer prevention for the next generation. J Adolesc Health. 2013; 52(5 suppl):S1–S7.
- 9. Centers for Disease Control and Prevention. [Accessed August 11, 2015] Cancer prevention among youth. Published 2015. Available at: www.cdc.gov/cancer/dcpc/prevention/youth.htm
- Centers for Disease Control and Prevention. [Accessed August 11, 2015] Cancer prevention during midlife. Published 2015 Available at: www.cdc.gov/cancer/dcpc/prevention/midlife.htm
- 11. Anderson LM, Diwan BA, Fear NT, Roman E. Critical windows of exposure for children's health: cancer in human epidemiological studies and neoplasms in experimental animal models. Environ Health Perspect. 2000; 108(suppl 3):573–594. [PubMed: 10852857]
- 12. Grandjean P, Barouki R, Bellinger DC, et al. Life-long implications of developmental exposure to environmental stressors: new perspectives. Endocrinology. 2015; 156(10):3408–3415. [PubMed: 26241067]
- 13. Interagency Breast Cancer and Environmental Research Coordinating Committee. Breast cancer and the environment: prioritizing prevention. Research Triangle Park, NC: National Institute of Environmental Health Sciences; 2013.
- Forman MR, Winn DM, Collman GW, Rizzo J, Birnbaum LS. Environmental exposures, breast development and cancer risk: through the looking glass of breast cancer prevention. Reprod Toxicol. 2015; 54:6–10. [PubMed: 25499721]
- 15. Metayer C, Dahl GV, Wiemels JL, Miller M. Childhood leukemia: a preventable disease. Pediatrics. 2016; 138(suppl 1)
- 16. Massetti GM, Thomas CC, Ragan KR. Disparities in the context of opportunities for cancer prevention in early life. Pediatrics. 2016; 138(suppl 1)
- 17. Holman DM, Ports KA, Buchanan ND, et al. The association between adverse childhood experiences and risk for cancer in adulthood: a systematic review of the literature. Pediatrics. 2016; 138(suppl 1)
- Latino-Martel P, Chan DS, Druesne-Pecollo N, Barrandon E, Hercberg S, Norat T. Maternal alcohol consumption during pregnancy and risk of childhood leukemia: systematic review and meta-analysis. Cancer Epidemiol Biomarkers Prev. 2010; 19(5):1238–1260. [PubMed: 20447918]
- Hargreave M, Jensen A, Deltour I, Brinton LA, Andersen KK, Kjaer SK. Increased risk for cancer among offspring of women with fertility problems. Int J Cancer. 2013; 133(5):1180–1186.
 [PubMed: 23404395]
- 20. McLaughlin CC, Baptiste MS, Schymura MJ, Nasca PC, Zdeb MS. Birth weight, maternal weight and childhood leukaemia. Br J Cancer. 2006; 94(11):1738–1744. [PubMed: 16736025]
- Biro FM, Wien M. Childhood obesity and adult morbidities. Am J Clin Nutr. 2010; 91(5):14998– 1505S. [PubMed: 20335542]
- 22. Caughey RW, Michels KB. Birth weight and childhood leukemia: a meta-analysis and review of the current evidence. Int J Cancer. 2009; 124(11):2658–2670. [PubMed: 19173295]
- 23. Paltiel O, Tikellis G, Linet M, et al. International Childhood Cancer Cohort Consortium.

 Birthweight and childhood cancer: preliminary findings from the International Childhood Cancer Cohort Consortium (I4C). Paediatr Perinat Epidemiol. 2015; 29(4):335–345. [PubMed: 25989709]
- 24. Milne E, Greenop KR, Metayer C, et al. Fetal growth and childhood acute lymphoblastic leukemia: findings from the Childhood Leukemia International Consortium. Int J Cancer. 2013; 133(12): 2968–2979. [PubMed: 23754574]
- 25. Xu X, Dailey AB, Peoples-Sheps M, Talbott EO, Li N, Roth J. Birth weight as a risk factor for breast cancer: a meta-analysis of 18 epidemiological studies. J Womens Health (Larchmt). 2009; 18(8):1169–1178. [PubMed: 19630536]

26. Cook MB, Akre O, Forman D, Madigan MP, Richiardi L, McGlynn KA. A systematic review and meta-analysis of perinatal variables in relation to the risk of testicular cancer—experiences of the son. Int J Epidemiol. 2010; 39(6):1605–1618. [PubMed: 20660640]

- 27. Zhou Y, Zhang S, Li Z, et al. Maternal benzene exposure during pregnancy and risk of childhood acute lymphoblastic leukemia: a meta-analysis of epidemiologic studies. PLoS One. 2014; 9(10):e110466. [PubMed: 25333868]
- 28. Soto AM, Vandenberg LN, Maffini MV, Sonnenschein C. Does breast cancer start in the womb? Basic Clin Pharmacol Toxicol. 2008; 102(2):125–133. [PubMed: 18226065]
- 29. Rezg R, El-Fazaa S, Gharbi N, Mornagui B. Bisphenol A and human chronic diseases: current evidences, possible mechanisms, and future perspectives. Environ Int. 2014; 64:83–90. [PubMed: 24382480]
- 30. Heck JE, Park AS, Qiu J, Cockburn M, Ritz B. An exploratory study of ambient air toxics exposure in pregnancy and the risk of neuroblastoma in offspring. Environ Res. 2013; 127:1–6. [PubMed: 24139061]
- 31. Verloop J, van Leeuwen FE, Helmerhorst TJ, van Boven HH, Rookus MA. Cancer risk in DES daughters. Cancer Causes Control. 2010; 21(7):999–1007. [PubMed: 20204493]
- 32. Troisi R, Hatch EE, Titus-Ernstoff L, et al. Cancer risk in women prenatally exposed to diethylstilbestrol. Int J Cancer. 2007; 121(2):356–360. [PubMed: 17390375]
- 33. Troisi R, Hatch EE, Titus L. The diethylstilbestrol legacy: a powerful case against intervention in uncomplicated pregnancy. Pediatrics. 2016; 138(suppl 1)
- 34. Bailey HD, Fritschi L, Metayer C, et al. Parental occupational paint exposure and risk of childhood leukemia in the offspring: findings from the Childhood Leukemia International Consortium. Cancer Causes Control. 2014; 25(10):1351–1367. [PubMed: 25088805]
- 35. Huoi C, Olsson A, Lightfoot T, et al. Parental occupational exposure and risk of childhood central nervous system tumors: a pooled analysis of case-control studies from Germany, France, and the UK. Cancer Causes Control. 2014; 25(12):1603–1613. [PubMed: 25281325]
- 36. Peters S, Glass DC, Greenop KR, et al. Childhood brain tumours: associations with parental occupational exposure to solvents. Br J Cancer. 2014; 111(5):998–1003. [PubMed: 24960405]
- 37. Bailey HD, Metayer C, Milne E, et al. Home paint exposures and risk of childhood acute lymphoblastic leukemia: findings from the Childhood Leukemia International Consortium. Cancer Causes Control. 2015; 26(9):1257–1270. [PubMed: 26134047]
- 38. Van Maele-Fabry G, Lantin AC, Hoet P, Lison D. Residential exposure to pesticides and childhood leukaemia: a systematic review and meta-analysis. Environ Int. 2011; 37(1):280–291. [PubMed: 20889210]
- Vinson F, Merhi M, Baldi I, Raynal H, Gamet-Payrastre L. Exposure to pesticides and risk of childhood cancer: a meta-analysis of recent epidemiological studies. Occup Environ Med. 2011; 68(9):694–702. [PubMed: 21606468]
- 40. Bailey HD, Fritschi L, Infante-Rivard C, et al. Parental occupational pesticide exposure and the risk of childhood leukemia in the offspring: findings from the Childhood Leukemia International Consortium. Int J Cancer. 2014; 135(9):2157–2172. [PubMed: 24700406]
- 41. Bailey HD, Infante-Rivard C, Metayer C, et al. Home pesticide exposures and risk of childhood leukemia: findings from the Childhood Leukemia International Consortium. Int J Cancer. 2015; 137(11):2644–2663. [PubMed: 26061779]
- 42. Ward MH, Colt JS, Deziel NC, et al. Residential levels of polybrominated diphenyl ethers and risk of childhood acute lymphoblastic leukemia in California. Environ Health Perspect. 2014; 122(10): 1110–1116. [PubMed: 24911217]
- 43. Ward MH, Colt JS, Metayer C, et al. Residential exposure to polychlorinated biphenyls and organochlorine pesticides and risk of childhood leukemia. Environ Health Perspect. 2009; 117(6): 1007–1013. [PubMed: 19590698]
- 44. Deziel NC, Rull RP, Colt JS, et al. Polycyclic aromatic hydrocarbons in residential dust and risk of childhood acute lymphoblastic leukemia. Environ Res. 2014; 133:388–395. [PubMed: 24948546]
- 45. Liu R, Zhang L, McHale CM, Hammond SK. Paternal smoking and risk of childhood acute lymphoblastic leukemia: systematic review and meta-analysis. J Oncol. 2011; 2011:854584. [PubMed: 21765828]

46. International Agency for Research on Cancer. A Review of Human Carcinogens: Personal Habits and Indoor Combustions. Lyon, France: WHO Press; 2012.

- 47. Antonopoulos CN, Sergentanis TN, Papadopoulou C, et al. Maternal smoking during pregnancy and childhood lymphoma: a meta-analysis. Int J Cancer. 2011; 129(11):2694–2703. [PubMed: 21225624]
- 48. Olivo-Marston SE, Yang P, Mechanic LE, et al. Childhood exposure to secondhand smoke and functional mannose binding lectin polymorphisms are associated with increased lung cancer risk. Cancer Epidemiol Biomarkers Prev. 2009; 18(12):3375–3383. [PubMed: 19959685]
- 49. Heck JE, Wu J, Lombardi C, et al. Childhood cancer and traffic-related air pollution exposure in pregnancy and early life. Environ Health Perspect. 2013; 121(11–12):1385–1391. [PubMed: 24021746]
- Boothe VL, Boehmer TK, Wendel AM, Yip FY. Residential traffic exposure and childhood leukemia: a systematic review and meta-analysis. Am J Prev Med. 2014; 46(4):413–422.
 [PubMed: 24650845]
- Danysh HE, Mitchell LE, Zhang K, Scheurer ME, Lupo PJ. Traffic-related air pollution and the incidence of childhood central nervous system tumors: Texas, 2001–2009. Pediatr Blood Cancer. 2015; 62(9):1572–1578. [PubMed: 25962758]
- 52. Xavier AC, Taub JW. Acute leukemia in children with Down syndrome. Haematologica. 2010; 95(7):1043–1045. [PubMed: 20595099]
- 53. Toledano SR, Lange BJ. Ataxia-telangiectasia and acute lymphoblastic leukemia. Cancer. 1980; 45(7):1675–1678. [PubMed: 6929216]
- 54. Inskip HM, Kinlen LJ, Taylor AM, Woods CG, Arlett CF. Risk of breast cancer and other cancers in heterozygotes for ataxia-telangiectasia. Br J Cancer. 1999; 79(7–8):1304–1307. [PubMed: 10098776]
- Schneider, K., Zelley, K., Nichols, KE., Garber, J., et al. Li-Fraumeni syndrome. In: Pagon, RA.Adam, MP., Ardinger, HH., editors. GeneReviews(R). Seattle, WA: University of Washington; 1993
- 56. Tiwari AK, Roy HK, Lynch HT. Lynch syndrome in the 21st century: clinical perspectives. QJM. 2016; 109(3):151–158. [PubMed: 26224055]
- 57. Thakker RV. Multiple endocrine neoplasia type 1 (MEN1) and type 4 (MEN4). Mol Cell Endocrinol. 2014; 386(1–2):2–15. [PubMed: 23933118]
- 58. Lip SZ, Murchison LE, Cullis PS, Govan L, Carachi R. A meta-analysis of the risk of boys with isolated cryptorchidism developing testicular cancer in later life. Arch Dis Child. 2013; 98(1):20–26. [PubMed: 23193201]
- 59. Spector LG, Pankratz N, Marcotte EL. Genetic and nongenetic risk factors for childhood cancer. Pediatr Clin North Am. 2015; 62(1):11–25. [PubMed: 25435109]
- 60. Park SK, Kang D, McGlynn KA, et al. Intrauterine environments and breast cancer risk: metaanalysis and systematic review. Breast Cancer Res. 2008; 10(1):R8. [PubMed: 18205956]
- 61. Barker DJ, Thornburg KL. Placental programming of chronic diseases, cancer and lifespan: a review. Placenta. 2013; 34(10):841–845. [PubMed: 23916422]
- 62. Biro FM, Deardorff J. Identifying opportunities for cancer prevention during preadolescence and adolescence: puberty as a window of susceptibility. J Adolesc Health. 2013; 52(suppl 5):S15–S20. [PubMed: 23601607]
- 63. Kamiya K, Ozasa K, Akiba S, et al. Long-term effects of radiation exposure on health. Lancet. 2015; 386(9992):469–478. [PubMed: 26251392]
- 64. Calvente I, Fernandez MF, Villalba J, Olea N, Nuñez MI. Exposure to electromagnetic fields (non-ionizing radiation) and its relationship with childhood leukemia: a systematic review. Sci Total Environ. 2010; 408(16):3062–3069. [PubMed: 20451240]
- 65. Jansen-van der Weide MC, Greuter MJ, Jansen L, Oosterwijk JC, Pijnappel RM, de Bock GH. Exposure to low-dose radiation and the risk of breast cancer among women with a familial or genetic predisposition: a meta-analysis. Eur Radiol. 2010; 20(11):2547–2556. [PubMed: 20582702]
- 66. The Surgeon General's Call to Action to Prevent Skin Cancer. Washington, DC: US Department of Health and Human Services, Office of the Surgeon General; 2014.

67. White MK, Pagano JS, Khalili K. Viruses and human cancers: a long road of discovery of molecular paradigms. Clin Microbiol Rev. 2014; 27(3):463–481. [PubMed: 24982317]

- 68. Ishaq S, Nunn L. Helicobacter pylori and gastric cancer: a state of the art review. Gastroenterol Hepatol Bed Bench. 2015; 8(suppl 1):S6–S14. [PubMed: 26171139]
- 69. Rusan M, Li YY, Hammerman PS. Genomic landscape of human papillomavirus-associated cancers. Clin Cancer Res. 2015; 21(9):2009–2019. [PubMed: 25779941]
- 70. Dahl S, Schmidt LS, Vestergaard T, Schüz J, Schmiegelow K. Allergy and the risk of childhood leukemia: a meta-analysis. Leukemia. 2009; 23(12):2300–2304. [PubMed: 19657364]
- 71. Linabery AM, Jurek AM, Duval S, Ross JA. The association between atopy and childhood/adolescent leukemia: a meta-analysis. Am J Epidemiol. 2010; 171(7):749–764. [PubMed: 20228139]
- 72. Heck JE, Ritz B, Hung RJ, Hashibe M, Boffetta P. The epidemiology of neuroblastoma: a review. Paediatr Perinat Epidemiol. 2009; 23(2):125–143. [PubMed: 19159399]
- Dikalioti SK, Chang ET, Dessypris N, et al. Allergy-associated symptoms in relation to childhood non-Hodgkin's as contrasted to Hodgkin's lymphomas: a case-control study in Greece and metaanalysis. Eur J Cancer. 2012; 48(12):1860–1866. [PubMed: 22230747]
- 74. Zhao H, Cai W, Su S, Zhi D, Lu J, Liu S. Allergic conditions reduce the risk of glioma: a meta-analysis based on 128,936 subjects. Tumour Biol. 2014; 35(4):3875–3880. [PubMed: 24347487]
- 75. Maisonneuve P, Lowenfels AB. Risk factors for pancreatic cancer: a summary review of metaanalytical studies. Int J Epidemiol. 2015; 44(1):186–198. [PubMed: 25502106]
- 76. Baer HJ, Tworoger SS, Hankinson SE, Willett WC. Body fatness at young ages and risk of breast cancer throughout life. Am J Epidemiol. 2010; 171(11):1183–1194. [PubMed: 20460303]
- 77. Martin RM, Gunnell D, Owen CG, Smith GD. Breast-feeding and childhood cancer: a systematic review with metaanalysis. Int J Cancer. 2005; 117(6):1020–1031. [PubMed: 15986434]
- 78. Saddlemire S, Olshan AF, Daniels JL, Breslow NE, Bunin GR, Ross JA. Breast-feeding and Wilms tumor: a report from the Children's Oncology Group. Cancer Causes Control. 2006; 17(5):687–693. [PubMed: 16633916]
- 79. Martin RM, Middleton N, Gunnell D, Owen CG, Smith GD. Breast-feeding and cancer: the Boyd Orr cohort and a systematic review with meta-analysis. J Natl Cancer Inst. 2005; 97(19):1446–1457. [PubMed: 16204694]
- 80. de Klerk N, Milne E. Overview of recent studies on childhood leukaemia, intra-uterine growth and diet. Radiat Prot Dosimetry. 2008; 132(2):255–258. [PubMed: 19124554]
- 81. Tower RL, Spector LG. The epidemiology of childhood leukemia with a focus on birth weight and diet. Crit Rev Clin Lab Sci. 2007; 44(3):203–242. [PubMed: 17453918]
- 82. Metayer C, Milne E, Dockerty JD, et al. Maternal supplementation with folic acid and other vitamins and risk of leukemia in offspring: a Childhood Leukemia International Consortium study. Epidemiology. 2014; 25(6):811–822. [PubMed: 25207954]
- 83. Rudant J, Lightfoot T, Urayama KY, et al. Childhood acute lymphoblastic leukemia and indicators of early immune stimulation: a Childhood Leukemia International Consortium study. Am J Epidemiol. 2015; 181(8):549–562. [PubMed: 25731888]
- 84. Ma X, Urayama K, Chang J, Wiemels JL, Buffler PA. Infection and pediatric acute lymphoblastic leukemia. Blood Cells Mol Dis. 2009; 42(2):117–120. [PubMed: 19064328]
- 85. Okasha M, McCarron P, Gunnell D, Smith GD. Exposures in childhood, adolescence and early adulthood and breast cancer risk: a systematic review of the literature. Breast Cancer Res Treat. 2003; 78(2):223–276. [PubMed: 12725422]
- 86. Kriebel D. The reactionary principle: inaction for public health. Occup Environ Med. 2007; 64(9): 573–574. [PubMed: 17704201]
- 87. Kriebel D. Cancer prevention through a precautionary approach to environmental chemicals. Rev Environ Health. 2009; 24(4):271–277. [PubMed: 20384033]
- 88. Agency for Toxic Substances and Disease Registry. [Accessed August 20, 2015] ATSDR toxic substances portal. Published 2015. Available at: www.atsdr.cdc.gov/substances/index.asp

89. Rooney AA, Boyles AL, Wolfe MS, Bucher JR, Thayer KA. Systematic review and evidence integration for literature-based environmental health science assessments. Environ Health Perspect. 2014; 122(7):711–718. [PubMed: 24755067]

- 90. Woodruff TJ, Sutton P. Navigation Guide Work Group. An evidence-based medicine methodology to bridge the gap between clinical and environmental health sciences. Health Aff (Millwood). 2011; 30(5):931–937. [PubMed: 21555477]
- 91. Woodruff TJ, Sutton P. The Navigation Guide systematic review methodology: a rigorous and transparent method for translating environmental health science into better health outcomes. Environ Health Perspect. 2014; 122(10):1007–1014. [PubMed: 24968373]
- 92. Genkinger JM, Terry MB. Commentary: towards a definite coherent heterogeneity in metaanalyses. Int J Epidemiol. 2014; 43(4):1236–1239. [PubMed: 24997208]
- 93. Wang SF, Shu L, Sheng J, et al. Birth weight and risk of coronary heart disease in adults: a metaanalysis of prospective cohort studies. J Dev Orig Health Dis. 2014; 5(6):408–419. [PubMed: 25263759]
- 94. Trasande L. Economics of children's environmental health. Mt Sinai J Med. 2011; 78(1):98–106. [PubMed: 21259266]
- 95. UMass Lowell Toxics Use Reduction Institute. Opportunities for cancer prevention: trends in the use and release of carcinogens in Massachusetts. Lowell, MA: University of Massachusetts Lowell; 2013.
- 96. Kriebel D, Hoppin PJ, Jacobs MM. Environmental and economic strategies for primary prevention of cancer in early life. Pediatrics. 2016; 138(suppl 1)
- 97. Erler C, Novak J. Bisphenol A exposure: human risk and health policy. J Pediatr Nurs. 2010; 25(5): 400–407. [PubMed: 20816563]
- 98. Bittner GD, Yang CZ, Stoner MA. Estrogenic chemicals often leach from BPA-free plastic products that are replacements for BPA-containing polycarbonate products. Environ Health. 2014; 13(1):41. [PubMed: 24886603]
- 99. California Environmental Protection Agency. [Accessed August 20, 2015] Ambient air quality monitoring. Published 2015. Available at: www.arb.ca.gov/aaqm/aaqm.htm
- 100. Centers for Disease Control and Prevention. [Accessed August 11, 2015] National Program of Cancer Registries. Published 2015. Available at: www.cdc.gov/cancer/npcr/
- 101. Centers for Disease Control and Prevention. [Accessed August 14, 2015] National Health and Nutrition Examination Survey. Published 2015. Available at: www.cdc.gov/nchs/nhanes.htm
- 102. Wong IO, Cowling BJ, Law SC, Mang OW, Schooling CM, Leung GM. Understanding sociohistorical imprint on cancer risk by age-period-cohort decomposition in Hong Kong. J Epidemiol Community Health. 2010; 64(7):596–603. [PubMed: 19710042]
- 103. Calafat AM, Longnecker MP, Koch HM, et al. Optimal exposure biomarkers for nonpersistent chemicals in environmental epidemiology. Environ Health Perspect. 2015; 123(7):A166–A168. [PubMed: 26132373]
- 104. Rozek LS, Dolinoy DC, Sartor MA, Omenn GS. Epigenetics: relevance and implications for public health. Annu Rev Public Health. 2014; 35:105–122. [PubMed: 24641556]
- 105. Intergovernmental Panel on Climate Change. [Accessed August 11, 2015] Home page. Available at: www.ipcc.ch/
- 106. Sutton P, Woodruff TJ. Risk communication and decision tools for children's health protection. Birth Defects Res C Embryo Today. 2013; 99(1):45–49. [PubMed: 23723171]
- 107. Commonwealth of Massachusetts Department of Public Health. Strategic plan for asthma in Massachusetts, 2015–2020. Boston, MA: Massachusetts Department of Health and Human Services; 2015.
- 108. Centers for Disease Control and Prevention. [Accessed August 12, 2015] National Comprehensive Cancer Control Program. Published 2015. Available at: www.cdc.gov/cancer/ncccp/index.htm
- 109. Reagan-Steiner S, Yankey D, Jeyarajah J, et al. National, regional, state, and selected local area vaccination coverage among adolescents aged 13–17 years—United States, 2014. MMWR Morb Mortal Wkly Rep. 2015; 64(29):784–792. [PubMed: 26225476]

110. Williams PRD. An analysis of violations of OSHA's (1987) occupational exposure to benzene standard. J Toxicol Environ Health B Crit Rev. 2014; 17(5):259–283. [PubMed: 25205215]

- 111. McHale CM, Zhang L, Smith MT. Current understanding of the mechanism of benzene-induced leukemia in humans: implications for risk assessment. Carcinogenesis. 2012; 33(2):240–252. [PubMed: 22166497]
- 112. Miglioretti DLJE, Johnson E, Williams A, et al. The use of computed tomography in pediatrics and the associated radiation exposure and estimated cancer risk. JAMA Pediatr. 2013; 167(8): 700–707. [PubMed: 23754213]
- 113. Terry MB, Bradbury A. Family-based breast cancer prevention efforts in adolescence. Pediatrics. 2016; 138(suppl 1)
- 114. Mackenzie A, Patrick-Miller L, Bradbury AR. Controversies in communication of genetic risk for hereditary breast cancer. Breast J. 2009; 15(suppl 1):S25–S32. [PubMed: 19775327]
- 115. Bradbury AR, Patrick-Miller L, Egleston BL, et al. Knowledge and perceptions of familial and genetic risks for breast cancer risk in adolescent girls. Breast Cancer Res Treat. 2012; 136(3): 749–757. [PubMed: 23065030]
- 116. Engel GL. The need for a new medical model: a challenge for biomedicine. Science. 1977; 196(4286):129–136. [PubMed: 847460]
- 117. Colditz GABK, Bohlke K, Berkey CS. Breast cancer risk accumulation starts early: prevention must also. Breast Cancer Res Treat. 2014; 145(3):567–579. [PubMed: 24820413]
- 118. Centers for Disease Control and Prevention. [Accessed August 12, 2015] NCHS Research Data Center. Published 2015. Available at: www.cdc.gov/rdc/
- Centers for Disease Control and Prevention. [Accessed August 12, 2015] Cancer data access for research. Published 2015. Available at: www.cdc.gov/cancer/npcr/data_access/
- 120. US Department of Health and Human Services. [Accessed August 17, 2015] Enhancing cancer registries for early case capture (ECC) of pediatric and young adult cancer (PYAC) cases. HHS grants forecast. Published 2014. Available at: www.acf.hhs.gov/hhsgrantsforecast/hhs_m/index.cfm?switch=grant.view&gff_grants_forecastInfoID=68002
- 121. Centers for Disease Control and Prevention. [Accessed August 12, 2015] Promoting cancer prevention. Published 2015. Available at: www.cdc.gov/cancer/promoting_prevention.htm

TABLE 1

Priori Coding Categories Used for the Qualitative Analysis of Meeting Transcripts

1. What is important?

Risk factors

For pediatric cancers

For cancers in adulthood

Protective factors

For pediatric cancers

For cancers in adulthood

Evidence needed to justify taking public health action

2. What is missing?

Additional data that would have the greatest impact or create tipping points for action

3. What can be done now?

The role of public health agencies

Lessons learned from other prevention efforts

Barriers to putting scientific findings into public health practice

Coordinating health promotion/disease prevention efforts toward broader shared goals

Communicating certainty or uncertainty to policy makers and the public

Avoiding unintended consequences

4. Other

A code for information not captured by the categories above

TABLE 2Examples of Early Life Factors Associated With Risk or Prevention of Pediatric and Adult Cancers

	Associated Cancers
Risk factors	
Adverse childhood events (eg, child abuse)	Adult cancers ¹⁷
Maternal alcohol consumption during pregnancy	Leukemia ¹⁸
Assisted reproductive technology	Pediatric cancers ¹⁹
Body weight	
Maternal prepregnancy weight	Pediatric cancers (eg, acute myeloid leukemia) ²⁰
Childhood obesity	Adult cancers (eg, esophageal adenocarcinoma) 21
High birth weight	Pediatric (eg, leukemia) ^{22–24} and adult (eg, breast cancer, ²⁵ testicular cancer ²⁶) cancers
Chemical agents	
Benzene	Childhood acute lymphoblastic leukemia ²⁷
BPA	Adult cancers ^{28,29}
Carbon tetrachloride	Neuroblastoma ³⁰
Diethylstilbestrol (prenatal exposure)	Adult cancers (eg, clear cell adenocarcinoma of the vagina and cervix) 31-33
Occupational exposures (paints, metals, hydrocarbons)	Pediatric cancers (eg, leukemia, brain and central nervous system tumors) $^{34-37}$
Pesticides, herbicides, and insecticides	Pediatric cancers (eg, leukemia, lymphoma, and brain cancer) $^{38-41}$
Polybrominated diphenyl ethers	Leukemia ⁴²
Polychlorinated biphenyl	Leukemia ⁴³
Polycyclic aromatic hydrocarbons	Leukemia, ⁴⁴ neuroblastoma ³⁰
Tobacco exposure	Pediatric (eg, leukemia, 45 hepatoblastoma, 46 non-Hodgkin lymphoma 47) and adult (eg, lung cancer 48) cancers
Traffic-related air pollution	Pediatric cancers (eg, leukemia) ^{49–51}
Chromosomal abnormalities (eg, Down syndrome)	Leukemia ⁵²
Genetic syndromes	
Ataxia telangiectasia	Pediatric (eg, leukemia) 53 and adult (eg, breast cancer) 54 cancers
Li-Fraumeni syndrome	Pediatric (eg, leukemia) and adult (eg, premenopausal breast cancer) 55 cancers $$
Lynch syndrome	Colorectal cancer ⁵⁶
Multiple endocrine neoplasia syndrome	Endocrine gland tumors, ⁵⁷ pediatric cancers
Isolated cryptorchidism	Testicular cancer ⁵⁸
Older parental age	Pediatric (eg, leukemia, lymphoma, neuroblastoma) 59 and adult (eg, breast cancer) 60 cancers
Placental phenotype (eg, extremely large or small)	Adult cancers ⁶¹
Pubertal events	
Early menarche	Adult cancers ⁶²
Age at peak height velocity	Adult cancers ⁶²
Radiation	

Ionizing radiation Pediatric (eg, leukemia) and adult (eg, thyroid and breast cancers) 63

Nonionizing radiation Pediatric (eg, leukemia) 64 and adult (eg, breast cancer) 65 cancers

UV radiation Skin cancers, ocular melanoma 66

Viruses and bacteria

Epstein-Barr virus Burkitt lymphoma, nasopharyngeal carcinoma 67

Herpesvirus Kaposi sarcoma 67 Helicobacter pylori Gastric cancer 68

Adult cancers (eg, cervical cancer) 67,69 Human papillomavirus

Protective and moderating factors

Childhood (eg, leukemia, 70,71 neuroblastoma, 72 non-Hodgkin Allergies, asthma, and atopy

lymphoma 73) and adult (eg, gliomas, 74 pancreatic cancer 75) cancers

Childhood obesity Breast cancer 21,76

Dietary factors

Breastfeeding Childhood (leukemia, lymphoma, Wilms tumor) 77,78 and adult (eg,

premenopausal breast cancer) 79 cancers

Pediatric cancers (eg, leukemia) 80-82 Maternal prenatal folic acid consumption Acute lymphoblastic leukemia 83,84

Early stimulation of the immune system (eg, day care attendance at younger age)

Physical activity during childhood Breast cancer 85

Note: This list is not exhaustive, and although some research findings suggest that these factors may be associated with subsequent cancer risk, the strength and consistency of the evidence varies. In addition, the types, sources, and quality of evidence (eg, animal models, observational studies, case-control studies, prospective cohort studies) vary as well.